pediatric annals

Commonly Asked Questions About Childhood Lead Poisoning

DEBORAH E. GLOTZER, MD, and MICHAEL WEITZMAN, MD

Does low-level lead poisoning cause neurocognitive impairment?

While there are individual studies that do not demonstrate cognitive impairments, the preponderance of evidence clearly indicates that low-level lead poisoniog causes neurocognitive impairment.[1-3] Meta-analysis, which analyzes data from multiple studies simultaneously, demonstrate adverse cognitive outcomes with elevated blood lead burdens, independent of confounding factors such as socioeconomic status.[2,3] The estimated average effect of an increase in a child's blood lead level is a decrease of 2 IQ points for each 10 ug/dL increase. However, the clinical significance for an individual child is uncertain. Some children may experience no measurable decreases and others may lose >2 IQ points for every 10 µg/dL increase in blood lead. Thus, the clinical significance for an individual child may be large, and when applied to a population of children, the significance is quite meaningful. It also should be noted that other more functional measures, such as behaviors measured by teacher rating scales, also are adversely affected by increasing lead burdens.[4] The behavioral manifestations of an elevated lead burden may be more noticeable than effects on intelligence.

The estimated average effect on an increase in a child's blood lead level is a decrease of 2 IQ points for each 10 µg/dL increase.

What are the primary sources of lead exposure in young children?

The almost complete elimination of lead from gasoline and canned foods plus its reduction in paints have reduced the incidence of elevated lead levels

Dr Glotzer is from Harvard University School of Medicine and The Cambridge Hospital, Cambridge, Massachusetts. **Dr Weitzman** is from University of Rochester School of Medicine and Dentistry, Rochester, New York. Address reprint requests to Deborah E. Glotzer, MD, The Cambridge Hospital, 1493 Cambridge St, Cambridge, MA 02139.

during childhood.[5,6] However, considerable lead exposure continues from other sources, and since lead is nonbiodegradeable, the impact of 60 years of use of leaded gasoline remains. The predominant sources of childhood lead poisoning are lead-based paint,[7,8] lead-contaminated household dust,[8-10] soil,[11] water,[8] household renovations,[8,12,13] improperly prepared infant formula,[8] occupational exposures with subsequent poisoning of children,[14] and lead-containing folk medicines and cosmetics.[15,16]

Lead-based paint is the predominant source of lead, and paint chips and lead-contaminated house dust are the primary vectors. This usually results from ingesting lead-based paint and lead-contaminated household dust during normal hand-to-mouth activity. Children living in pre-1960 homes, particularly those that have deteriorating surfaces, are most at risk. In contrast to toddlers and preschoolers, infants are more likely to have been poisoned during household renovations or from improper preparation of infant formula. However, all potential sources should be explored in a poisoned child.

What is the significance of the lowering of the threshold blood lead level considered elevated by the Centers for Disease Control and Prevention [CDC]?

In 1991, the CDC lowered the blood lead level considered to be elevated in children from the 25 ug/dL threshold established in 1985 to 10 µg/dL.[17] This resulted from accumulating data indicating adverse health effects with blood lead levels previously thought innocuous. It should be noted that the 1991 CDC guidelines did not apply the term "lead poisoned" to children with lead levels 25 ug/dL. The CDC has not advocated that children with lead levels between 10 and 25 ug/dL receive chelation or other services previously recommended for lead levels ≥25 ug/dL. Rather, these children should receive focused counseling to reduce lead exposure and follow-up screening to detect further increases in the blood lead level. Environmental investigation and remediation may be recommended by childhood lead poisoning prevention programs in some communities. Families of all children, including those with blood lead levels <10 ug/dL, should receive anticipatory guidance regarding the hazards and likely sources of lead exposure.

Does universal lead screening make sense?

In its 1991 guidelines, the CDC stated that "virtually all US children are at risk for lead poisoning," and recommended universal blood lead screening, except for children who live in a community that has demonstrated that it does not have a childhood lead poisoning problem.[17] The American Academy of Pediatrics (AAP) has taken a slightly less aggressive stance due to the concern that sufficient laboratory capacity does not currently exist for universal screening, but recommends that "blood lead screening should be a part of routine health supervision for children."[18] Furthermore, a number of states mandate blood lead screening. As screening data are analyzed, it is likely that there are locales where selective rather than universal blood lead screening is appropriate. Some have suggested that universal blood lead screening is not a wise use of financial resources, and it would be preferable to focus attention and resources to other childhood problems such as under-immunization, premature births, child abuse and violence.[19,20] These arguments are based on the premises that the adverse effects of low-level poisoning are clinically insignificant and that lead surveillance and management is costly, with marginal or unproven benefit. These issues can only be resolved with formal economic evaluations that examine the costs, benefits, and effectiveness of lead screening compared to other programs.

The almost complete elimination of lead from gasoline and canned foods plus its reduction in paints have reduced the incidence of elevated lead levels during childhood.

How effective are risk questionnaires in predicting elevated blood lead levels and the need for blood lead testing?

It has been suggested that risk questionnaires may help differentiate between children at high versus low risk for high-dose lead exposure.[17] In proposing that children can be classified as high or low risk, the CDC did not suggest that low risk children do not require screening but instead that the screening schedule is different depending on risk classification. Subsequent to the publication of the CDC guidelines, at least four groups of investigators have studied whether risk questionnaires are effective in identifying children with elevated blood lead levels (Table).[21-24]

Sensitivity indicates the percentage of children with blood lead levels ≥10 µg/dL who were classified as high-risk by questionnaire, ie, how well does the questionnaire identify children with elevated blood lead levels. The negative predictive value indicates the probability that a child has a blood lead level <10 µg/dL if the child is classified as low risk. The questionnaire is most sensitive and has a higher negative predictive value when the prevalence of elevated blood lead levels is lowest. In areas of moderate prevalence, the ability of the questionnaire to accurately identify children with elevated blood lead levels is diminished and should not be a substitute for blood lead screening.

TABLE

Effectiveness of Questionnaires in Determining Risk Classification

Study	Prevalence of Blood Lead Level ≥10 µg/dL (%)	Sensitivity (%)	Negative Predictive Value (%)
Schaffer et al	28.0	70.0	81.0
Binns et al	2.1	69.0	99.0
Tejeda et al	7.0	90.0	99.0
Rooney et al (clinic A)	5.4	76.9	96.5
Rooney et al (clinic B)	16.8	63.6	81.4

Should samples for lead screening be obtained by capillary or venous sample?

Initial screening for childhood lead poisoning is usually done by capillary sampling. However, capillary samples are more likely to be falsely elevated due to skin contamination than are venipuncture samples.[25] In addition, the smaller volume of blood from a capillary puncture further increases uncertainty. Therefore, the CDC recommends that a blood lead level ≥15 µg/dL from a capillary sample be confirmed by a venous sample. If the prevalence of elevated blood lead levels or the false positive rate of capillary samples is high, initial screening by venipuncture probably will be less expensive for initial screening.[26] The CDC has stated that "venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practicable."[17]

What advice regarding lead poisoning should be given to pregnant and nursing mothers?

Lead circulating in the maternal bloodstream is readily transferred to the fetus, and there is a high correlation between lead in maternal and umbilical cord blood.[27,28] Lead also is transferred from maternal stores into breast milk, but concentrations are lower than in maternal blood.[27] In addition, pregnancy may lead to mobilization of bone lead stores, secondary to changes in calcium metabolism and bone turnover associated with pregnancy.[28] Thus, it seems reasonable to counsel pregnant women to avoid lead exposure, and women who have known or suspected lead exposure, including poisoning during childhood, undergo blood lead screening prior to pregnancy or lactation. However, data are limited, and currently there are no official recommendations regarding lead screening in pregnant or lactating women.

What is the relationship between iron deficiency and elevated lead levels?

Although environmental lead exposure is the largest contributor to a child's risk of lead poisoning, lead kinetics and nutritional status also play roles in susceptibility to childhood lead poisoning. Lead absorption and retention is significantly higher in infants and young children than in older individuals.[29] Total food intake and dietary intake of zinc, calcium, and particularly iron influence susceptibility to lead toxicity.[30] Thus, susceptibility is compounded because children who are epidemiologically at highest risk for lead exposure are also more likely to have these nutritional deficiencies. Among the nutritional factors that appear to influence susceptibility to lead toxicity, iron status and intake are the most clinically important.

It is well demonstrated that iron deficiency and inadequate iron intake increase the susceptibility to lead intoxication. Iron-fortified foods during infancy can decrease iron deficiency.[31,32] The CDC recommends nutritional counseling for children with elevated lead burdens and iron supplementation for those with iron deficiency.[17] Prophylactic nutritional supplementation, especially iron, for children at high risk for nutritional deficiencies may reduce lead poisoning. However, the effectiveness of nutritional counseling or supplementation for preventing or treating lead poisoning has not been studied.

Are dietary interventions helpful in the management of a child with an elevated blood lead level?

While there are no clinical trials regarding nutritional interventions in children, data from animal studies and studies in human adults indicate nutritional interventions can reduce lead retention. These data indicate iron deficiency increases lead absorption, and adequate iron intake decreases lead absorption. Animal studies indicate lead retention is increased by calcium deficiency, and even in calcium-replete animals, calcium supplementation reduces iron retention, probably through competition of binding sites in the intestinal mucosa. Phosphorous intake also appears to moderate oral lead absorption. Finally, lead absorption is increased in the fasting state.[33]

Based on these data, the following simple and practical nutritional interventions may benefit children with elevated blood lead levels or children at

continued on page 637

continued from page 632
risk for excessive lead exposure: 1) evaluate for iron deficiency and treat with supplemental iron if deficient, 2) provide a diet with adequate iron, 3) avoid prolonged fasting by eating regular meals and snacks (three meals and two snacks for most toddlers and preschoolers), and 4) ensure adequate calcium intake. Although milk and other dairy products are excellent sources of calcium excess cow's milk intake must be avoided as this can lead to occult gastrointestinal blood loss and subseqent iron deficiency.

Should an EDTA mobilization test be conducted for a child with a blood lead level ≥25 µg/dL?

The 1991 CDC guidelines state that "many experienced practitioners decide whether to use chelation therapy on the basis of the results of carefully performed $CaNa^2EDTA$ mobilization tests (for children with blood lead levels of 25 to 44 µg/dL)."[17] The premise of the test is that children with a positive test are most likely to experience a significant lead diuresis from chelation therapy.[34,35] However, the probability of a positive test for a given blood lead level varies widely among centers.[34, 36] Therefore, clinicians may wish to base the lead level range for performing a mobilization test on the likelihood this will be positive at their own institution. A national survey of pediatric lead poisoning treatment centers, revealed considerable variability in the range of blood lead levels for which EDTA mobilization tests were recommended as the initial management.[37]

What interventions are effective for the treatment of children with elevated blood lead levels?

The most important intervention is preventing additional exposure. Although lead paint abatement often is viewed as an effective means of reducing a child's lead exposure, some deleading procedures can elevate blood lead levels.[13,38] Therefore, it is essential that parents of children with elevated lead burdens understand that lead paint removal requires strict safeguards, special equipment and training. Partial remediation frequently is recommended. One recent retrospective study in St Louis, Missouri investigated the impact of limited remediation on children with initial venous lead levels ≥25 µg/dL.[39] Blood lead declined more in children whose homes underwent remediation procedures compared with those whose homes did not, but the effect was negligible (mean decline of 1% due to remediation procedures) for children with blood lead levels between 25 and 34 µg/dL. In 1983, Charney et al[40] demonstrated that for children with moderate lead poisoning (lead levels 30 to 49 µg/dL), periodic dust-control measures in conjunction with lead paint reduction was more effective in reducing blood lead levels and the need for chelation than lead paint reduction alone. Dust control measures consisted of wet mopping all rooms with elevated dust lead

Lead-based paint is the predominant source of lead, and paint chips and lead-contaminated house dust are the primary vectors.

levels twice monthly with a high phosphate detergent. Parents also were instructed to wet mop hazardous areas two to three times per week and to wash their child's hands frequently. The most marked reductions in blood lead levels were seen in children with the highest initial values. Thus, the data suggest that environmental interventions are most effective for children with the highest blood lead levels and presumably the most lead in their environments. It is unlikely that dust-control measures and frequent hand washing are detrimental to any child so it appears prudent to recommend dust-control measures for children with elevated lead burdens.

What is the best chelating agent for a child with an elevated blood lead level?

There is no standard approach for treatment of childhood lead poisoning. Even centers that specialize in lead poisoning differ in recommendations for management of poisoned children.[37] Survey results indicate a wide range in the minimum blood lead level for which chelation is recommended (20 to 50 µg/dL) and a wide variety of recommended chelating agents. The 1991 CDC statement, "Preventing Lead Poisoning in Young Children," makes no specific recommendation regarding the treatment of asymptomatic children with levels of 25 to 44 µg/dL.[17] This lack of consensus is likely due to the lack of hard data regarding the impact of chelation on poisoned children.

Under what circumstances should an oral chelator be used?

Oral chelators permit treatment on an ambulatory basis. However, chelation is an adjunct, rather than a substitute for environmental remidiation, and the most important treatment of a lead-poisoned child is to prevent ongoing lead exposure. Thus, outpatient chelation should occur only if environmental remediation has occurred. The physician also must assess whether compliance and appropriate follow-up visits are likely to be satisfactory. Both penicillamine and DMSA have an unpleasant odor and taste, and are not available in a liquid formulation, so parents must be prepared to administer medication to unwilling children.

How effective is chelation in preventing adverse sequelae of elevated lead burdens?

Although it is well accepted that children with

The data suggest that environmental interventions are most effective for children with the highest blood lead levels and presumably the most lead in their environments.

lead encephalopathy or severe plumbism (≥100 µg/dL) benefit from chelation,[41] it is unclear if chelation therapy prevents or reduces developmental morbidity for children with less severe lead burdens. One recent study suggests there may be modest improvements in cognitive performance associated with reductions in blood lead levels, but the improvement was not specifically related to chelation.[42] However, non-nervous system functions, such as hematopoiesis, vitamin D metabolism, growth hormone and thyroid-stimulating hormone, are improved following chelation therapy.[41,43-46] These biochemical data, coupled with documented benefits of chelation in children with symptomatic lead poisoning, lead to the working hypothesis that chelation is likely to improve neurocognitive outcome in children with asymptomatic low-level poisoning. This premise forms the basis for many approaches for the management of children with elevated blood lead levels. However, uniform opinion regarding the optimal strategy does not exist.

Should children known to have elevated blood lead levels be permitted to remain in or visit homes known to contain lead-based paint?

All homes containing lead-based paint have the potential to cause significant lead exposure in children but the risk of exposure is related primarily to contact with lead-contaminated household dust and non-intact painted surfaces. Several studies demonstrate that housing condition is an important contributor to children's blood lead levels.[47,48] The likelihood that a child will sustain significant lead exposure is logically related to both the duration and intensity of exposure. Therefore, it makes sense to recommend that children not spend significant time, such as day care, in homes with deteriorating surfaces if they have lead-based paint.

What precautions should be taken before household renovations are performed?

The average content of lead in paint began to decrease in the 1950s and lead-based paint was banned for residential use in 1977. It is estimated that more than half of in the United States contain paint with lead concentrations >0.7 mg/cm2.[49] Therefore, it is prudent to document the lead content of any surfaces that will be disturbed during renovations in homes built before 1978, and particularly in those built before 1960. If any contain leaded paint, the occupants can consult with a trained deleader before disturbing surfaces. At a minimum, it makes sense to: 1) relocate young children and pregnant women from the premises until the renovations, and thorough clean-up with a high-phosphate cleaner (such as trisodium phosphate) is completed, and 2) use renovation methods that generate the least amount of dust and debris.

Should all homes known to contain lead-based paint be deleaded?

Although some have advocated deleading all homes containing lead-based paint, it is unclear whether this is the best use of financial resources for the following reasons. Remediation of lead paint-based household hazards is most effective for children with the highest blood lead levels.[39] Only meticulous and careful abatement is likely to have lasting beneficial effects on dust lead levels.[38] Thus, the highest priority for deleading is for children with the highest lead levels, and this should be done using methods that have been shown to cause a lasting reduction in dust lead levels. Such methods are likely to benefit current and future household occupants. If cost is not a factor, meticulous abatement for all lead-containing homes have the potential to benefit large numbers of children over time. The cost-benefit ratio of these procedures has not been established. It is likely that the ratio will vary with housing condition and blood lead level.

How can you tell if household water is the source of elevated blood lead?

Although water can contribute to a child's lead burden, it is not likely to cause a substantial increase except in unusual cases.[8] For infants whose total food and fluid intake comes from reconstituted formula and juice, lead in water contributes to the overall body lead burden to a greater extent than in older children and adults. Lead can enter drinking water from municipal water distribution systems or household plumbing if there are lead pipes or if lead-based solder was used. Lead can leach into drinking water if it has been standing in lead-containing plumbing systems. Therefore, if the water faucet has not been used for more than 6 hours (such as overnight), flush the system by letting tap water run for 15 to 30 seconds, or until there is a noticeable change in water temperature, before using the water for eating or drinking purposes; Use only cold water for cooking and drinking, as lead can dissolve more readily into hot water. Finally replace lead pipes if possible. A nationwide sampling of lead in household water, conducted by Consumers Union, found no detectable lead in first draw or fully flushed water of 61% and 90% of households, respectively.[50] Only 4% of households sampled had first draw water that exceed the US Environmental Pro-tection Agency action level of 15 parts per billion. Household water can be analyzed for

lead in commercial laboratories. The appearance of the water is not related to its lead content.

If soil lead levels are known to be elevated, what should be done?

While it is well established that lead-contaminated soil can contribute to the lead burdens of children with elevated blood lead levels,[10,11] soil abatement is not generally recommended in children with mild elevations.[11] A randomized clinical trial of children with blood lead of 7 to 24 µg/dL revealed that soil abatement resulted in a modest decline in blood lead.[11] However, the magnitude of the decrease was small. Because of the high cost of soil removal, it is not likely to be a useful clinical intervention for most children. There may be individual children for whom soil is the overwhelming contributor to a substantial lead burden, so the issue of soil removal should be evaluated on an individual basis.

Under what circumstances should acute lead intoxication be included in a differential diagnosis?

Acute lead intoxication can result in headaches, vomiting, constipation, severe abdominal cramps, ataxia, unconsciousness or seizures, and should be included in the differential diagnosis of the child with these problems if the diagnosis is not apparent. Any child who is symptomatic from acute lead poisoning represents a medical emergency and should be hospitalized.

REFERENCES

1. Bellinger D, Dietrich KN. Low-level lead exposure and cognitive function in children. *Pediatr Ann.* 1994;23:600-605.
2. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children: a meta-analysis of modern studies. *JAMA.* 1990;263:673-678.
3. Schwartz J. Societal benefits of reducing lead exposure. *Environ Res.* 1994;66:105-124.
4. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med.* 1979;300:689-695.
5. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988-1991). *JAMA.* 1994;272:277-283.
6. Pirkle JL, Brody DJ, Gunter EW, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA.* 1994;272:284-291.
7. McElvaine MD, DeUngria EG, Matte TD, Copley CG, Binder S. Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning, St Louis, Missouri, 1989 through 1990. *Pediatrics.* 1992;89:740-742.
8. Shannon MW,Graef JW. Lead intoxication in infancy. Pediatrics. 1992;89:87-90.
9. Sayre JW, Charney E, Vostal J, Pless IB. House and hand dust as a potential source of childhood lead exposure. *Am J Dis Child.* 1974; 127:167-170.
10. Charney E, Sayre J, Coulter M. Increased lead absorption in inner city children: where does the lead come from? Pediatrics. 1980;65:226-231.
11. Weitzman M, Aschengrau A, Bellinger D, Jones R, Hamlin JS, Beiser A. Lead-contaminated soil abatement and urban children's blood lead levels. *JAMA.* 1993;269:1647-1654.
12. Amitai Y, Graef JW, Brown MJ, Gerstle RS, Kahn N, Cochrane PE. Hazards of deleading homes of children with lead poisoning. *Am J Dis. Child.* 1987;141:758-760.
13. Rey-Alvarez S, Menke-Hargrave T. Deleading dilemma: pitfall in the management of childhood lead poisoning. *Pediatrics.* 1987;79:214-217.
14. Baker EL, Folland DS, Taylor TA, et al. Lead poisoning in children of lead workers: home contamination with industrial dust. N Engl J Med. 1977;296:260-261.
15. Saryan LA. Surreptitious lead exposure from an Asian-Indian medication. *J Analyt Toxicol.* 1991;15:336-338.
16. Nir A, Tamir A, Zelnik N, Iancu TC. Is eye cosmetic a source of lead poisoning? *Israel J Med Sci.* 1992;28:417-421.
17. Centers for Disease Control. Preventing Lead Poisoning in Young Children: A statement by the Centers for Disease Control. Atlanta, GA: US Dept of Health and Human Services; 1991.
18. American Academy of Pediatrics Committee on Environmental Health. Lead poisoning: from screening to primary prevention. *Pediatrics.* 1993;92:176-183.
19. Sayre JW, Ernhart CB. Control of lead exposure in childhood: are we doing it correctly? *Am J Dis Child.* 1992;146:1275-1278.
20. Schoen EJ. Childhood lead poisoning definitions and priorities. *Pediatrics.* 1993;91:504-505.
21. Binns HJ, LeBailly SA, Poncher J, Kinsella TR, Saunders SE, Pediatric Practice Research Group. Is there lead in the suburbs? Risk assessment in Chicago suburban pediatric practices. *Pediatrics.* 1994;93:164-171.
22. Rooney BL, Hayes EB, Allen BK, Strutt PJ. Development of a screening tool for prediction of children at risk for lead exposure in a midwestern clinical setting. *Pediatrics.* 1994;93:183.
23. Schaffer SJ, Szilagyi PG, Weitzman M. Lead poisoning risk determination in an urban population through the use of a standardized questionnaire. *Pediatrics.* 1994;93:159-163.
24. Tejeda DM, Wyatt DD, Rostek BR, Solomon WB. Do questions about lead exposure predict elevated lead levels? Pediatrics. 1994;93:192-194.
25. Schlenker TL, Fritz CJ, Mark D, et al. Screening for pediatric lead poisoning: comparability of simultaneously drawn capillary and venous blood samples. *JAMA.* 1994;271:1346-1348.
26. Glotzer DE, Bauchner H, Freedberg KA, Palfrey S. Screening for childhood lead poisoning: a cost-minimization analysis. *Am J Public Health.* 1994;84: 110-112.
27. Ong CN, Phoon WO, Law HY, Tye CY, Lim HH. Concentrations of lead in maternal blood, cord blood, and breast milk. *Arch Dis Child.* 1985;60:756-759.
28. Silbergeld EK. Lead in bone: implications for toxicology during pregnancy and lactation. *Environ Health Perspect.* 1991;91:63-70.
29. Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, Foman SJ. Absorption and retention of lead by infants. *Pediatr Res.* 1978;12:29-34.
30. Mahaffey KR. Nutritional factors in lead poisoning. *Nutri Rev.* 1981;39:353-362.
31. Pizarro F, Yip R, Dallman PR, Olivares M, Hertrampf E, Walter T. Iron status with different infant feeding regimens: relevance to screening and prevention of iron deficiency. *J Pediatr.* 1991;118:687-692.
32. Walter T, Dallman PR, Pizarro F, et al. Effectiveness of iron-fortified infant cereal in prevention of iron deficiency anemia. *Pediatrics.* 1993;91:976-982.
33. Rabinowitz MB, Kopple JD, Wetherill GW. Effect of food intake and fasting on gastrointestinal lead absorption in humans. *Am J Clin Nutr.* 1980;33:1784-1788.
34. Markowitz ME, Rosen JF. Need for the lead mobilization test in children with lead poisoning. J Pediatr. 1991;119:305-310.
35. Piomelli S, Rosen JF, Chisolm JJ Jr, Graef JW. Management of childhood lead poisoning. *J Pediatr.* 1984;105:523-532.
36. Weinberger HL, Post EM, Schneider T, Helu B, Friedman J. An analysis of 248 initial mobilization tests performed on an ambulatory basis. *Am J Dis Child.* 1987;141:1266-1270.
37. Glotzer DE, Bauchner H. Management of childhood lead poisoning: a survey. *Pediatrics.* 1992;89:614-618.
38. Farfel MR, Chisolm JJ. Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. Am J Public Health. 1990;80:1240-1245.
39. Staes C, Matte T, Copley CG, Flanders D, Binder S. Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels in St Louis, Missouri. *Am J Epidemiol.* 1994;139:1016-1026.
40. Charney E, Kessler B, Farfel M, Jackson D. Childhood lead poisoning. A controlled trial of the effect of dust-control measures on blood lead levels. *N Engl J Med.* 1983;309:1089-1093.
41. Chisolm JJ. The use of chelating agents in the treatment of acute and chronic lead intoxication in childhood. *J Pediatr.* 1968;73:1-38.
42. Ruff HA, Bijur PE, Markowitz M, Ma YC, Rosen JF. Declining blood lead levels and cognitive changes in moderately lead-poisoned children. *JAMA.* 1993;269:1641-1646.
43. Chisolm JJ Jr. Heme metabolites in blood and urine in relation to lead toxicity and their determination. *Adv Clin Chem.* 1978;20:225-265.
44. Rosen JF, Chesney RW, Hamstra A, DeLuca HF, Mahaffey KR. Reduction in 1,25-dihydroxy vitamin D in children with increased lead absorption. *N Engl J Med.* 1980;302:1128-1131.
45. Huseman CA, Varma MM, Angle CR. Neuroendocrine effects of toxic and low blood lead levels in children. *Pediatrics.* 1992;90:186-189.
46. Huseman CA, Moriarty CM, Angle CA. Childhood lead toxicity and impaired release of thyrotropin-stimulating hormone. Environ Res. 1987;42:524-533.
47. Chisolm JJ, Mellits ED, Quaskey SA. The relationship between the level of lead absorption in children and the age, type, and condition of housing. *Environ Res.* 1985;38:3145.
48. Clark CS, Bornschein RL, Succop P, Que Hee SS, Hammond PB, Peace B. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res.* 1985;38:46-53.
49. Agency for Toxic Substances and Disease Registry. *The Nature and Extent of Lead Poisoning in the United States: A Report to Congress.* Atlanta, US Dept of Health and Human Services, Public Health Service; 1988.
50. Is there lead in your water? *Consumer Reports.* February 1993:73-82.